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Correlation Of Diabetic Retinopathy Severity With Myocardial Remodeling And Quality Of Life In Type II Diabetes Mellitus Patients

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Abstract

Background*: Type II diabetes mellitus (T2DM) is associated with microvascular complications like diabetic retinopathy (DR) and macrovascular complications such as myocardial remodeling, both of which impair quality of life (QoL). This study explores the relationship between DR severity, cardiac structural changes, and QoL in T2DM patients to guide integrated care.*

Methods*: A cross-sectional study was conducted at R. L. Jalappa Hospital, Kolar, involving 97 T2DM patients with over 5 years of disease duration and no prior cardiac history. Assessments included visual acuity (Snellen chart), slit-lamp biomicroscopy, posterior segment evaluation (indirect ophthalmoscopy, +90D biomicroscopy), echocardiography, and electrocardiography (ECG). DR was graded using the Early Treatment Diabetic Retinopathy Study (ETDRS) criteria. QoL was evaluated with the using Cardiomyopathy Questionnaire (Kansas city)- KCCQ-12. Data were analyzed using SPSS, employing descriptive statistics and logistic regression ($p<0.05$).*

Results*: DR severity correlated with increased left ventricular end-diastolic diameter (LVDD) (47.3±6.5 mm in proliferative DR [PDR] vs. 42.5±4.2 mm in normal fundus, $p=0.01$), left ventricular systolic diameter (LVDS) (33.2±5.7 mm vs. 28.3±3.8 mm, $p=0.02$), and reduced left ventricular ejection fraction (LVEF) (43.5±8.9% vs. 55.2±6.1%, $p<0.001$). ECG abnormalities were prevalent in 90% of PDR patients ($p<0.001$). Severe DR was associated with elevated systolic blood pressure (135.4±18.6 mmHg vs. 124.3±12.8 mmHg, $p=0.03$), poor glycemic control (HbA1c 10.8±2.5% vs. 8.1±1.6%, $p<0.001$), and nephropathy markers (serum creatinine 2.4±1.5 mg/dL vs. 1.2±0.6 mg/dL, $p<0.01$). QoL declined significantly with DR progression ($p=0.04$), driven by cardiac symptoms and psychological distress. Diabetes duration >10 years was linked to severe DR, cardiac remodelling, and renal impairment ($p<0.001$).*

Conclusion*: Progressive DR is associated with myocardial remodelling, increased cardiovascular risk, and reduced QoL in T2DM patients. Severe DR serves as a marker for cardiac dysfunction, necessitating integrated ophthalmic and cardiovascular screening. Early glycemic control and multidisciplinary care are critical to mitigate complications and enhance QoL.*

Keywords*: Type II Diabetes Mellitus, Diabetic Retinopathy, Myocardial Remodelling, Quality of Life, Cardiovascular Risk, Multidisciplinary Care*

INTRODUCTION

Type II diabetes mellitus (T2DM) is a global health challenge characterized by chronic hyperglycaemia due to insulin resistance and beta-cell dysfunction[1]. T2DM leads to microvascular complications, such as diabetic retinopathy (DR), and macrovascular complications, including myocardial remodelling, both contributing to significant morbidity and mortality [2,3]. DR, a leading cause of blindness, results from hyperglycaemia-induced retinal microvascular damage, progressing from non-proliferative DR (NPDR) to proliferative DR (PDR) and diabetic macular edema (DME)[4]. Myocardial remodelling, a hallmark of diabetic cardiomyopathy, involves structural and functional cardiac changes, including left ventricular hypertrophy, fibrosis, and diastolic dysfunction, often progressing to heart failure[5].

Emerging evidence suggests shared pathophysiological pathways between DR and myocardial remodelling, including oxidative stress, inflammation, endothelial dysfunction, and renin-angiotensin-aldosterone system (RAAS) activation[6,7]. These mechanisms link retinal and cardiac microvascular damage, positioning DR as a potential indicator of cardiovascular risk [8]. Both conditions significantly impair quality of life (QoL) due to visual loss, cardiac symptoms, and psychological distress[9]. However, the direct correlation between DR severity, myocardial remodelling, and QoL remains underexplored. This study aims to analyse the association between DR severity and myocardial remodelling using echocardiography and ECG and to assess the association of grades of retinopathy with other diabetic

target organ damage (Diabetic nephropathy) and Quality of life using Cardiomyopathy Questionnaire (Kansas city)- KCCQ-12

MATERIALS AND METHODS

This cross-sectional observational study was conducted at R. L. Jalappa Hospital & Research Centre, Kolar, India, in collaboration with Narayana Health Heart Centre, over 18 months. Ethical approval was obtained from the Institutional Ethics Committee of Sri Devaraj Urs Medical College, and written informed consent was secured from all participants. Ninety-seven T2DM patients, diagnosed for over 5 years and without prior cardiac history, were recruited and those with retinopathy due to other causes like hypertension, anaemia, leukaemia, hypercoagulable states and radiation exposure were excluded.
Sample size was estimated by using the proportion of diabetic retinopathy in subjects who had DM was 65.3% from the study by Seyhmus kulahcioglu et al.[16] using the formula

$$\text{Sample Size} = \frac{Z1\text{-}\alpha/2\ 2\ P\ (1\text{-}P)}{d2}$$

$Z1\text{-}\alpha/2$ = is standard normal variate (at 5% type 1 error ($P<0.05$) it is 1.96 and at 1% type1 error ($P<0.01$) it is 2.58). As in majority of studies P values are considered significant below 0.05 hence 1.96 is used in formula. P= Expected proportion in population based on previous studies or pilot studies. d= Absolute error or precision. P = 65.3% or 0.653. q = 34.7 or 0.347
and = 10% or 0.10. Using the above values at 95% Confidence level a sample size of 88 subjects with Diabetes Mellitus will be included in the study. Considering 10% Nonresponse a sample size of 88 + 8.8 ≈ 97 subjects will be included in the study.
Patients meeting the inclusion criteria were enrolled and underwent a detailed clinical evaluation, including visual acuity assessment using a Snellen chart, slit lamp biomicroscopy for anterior segment examination, and posterior segment evaluation via indirect ophthalmoscopy and +90D biomicroscopy by two blinded ophthalmologists. Diabetic retinopathy was graded according to the Early Treatment Diabetic Retinopathy Study (ETDRS) classification. Blood investigations included fasting and postprandial blood sugar, HbA1c, urea, and creatinine levels. Cardiac assessment was performed using ECG and 2D echocardiography following American Society of Echocardiography (ASE) guidelines, evaluating left ventricular (LV) dimensions, volumes, ejection fraction (LVEF via Simpson's method), and left atrial size. Quality of life was assessed using the Kansas City Cardiomyopathy Questionnaire (KCCQ-12).

Statistical Analysis

Data were analysed using SPSS software. Descriptive statistics (means, standard deviations, proportions) summarized patient characteristics. Logistic regression models, adjusted for confounders (age, diabetes duration), explored associations between DR severity, cardiac remodelling, nephropathy, and QoL. Significance was set at $p<0.05$, with results reported as odds ratios and 95% confidence intervals.

RESULTS

Demographic analysis showed that NPDR was more prevalent among older participants, with the highest proportions in the 61–70 (31.6%) and >70 (26.3%) age groups, while it was rare in those <40 years (2.6%). Increasing severity of NPDR was more common with advancing age, and the age–severity association approached significance ($p = 0.05$). Gender distribution revealed a higher prevalence among males (57.9%) compared to females (42.1%), with moderate and severe NPDR more frequent in men, whereas women more often presented with mild NPDR; this difference was statistically significant ($p = 0.01$). [Table 1]

Variable		Mild NPDR	Moderate NPDR	Severe NPDR	Total	P value
Age group	<40	1 (2.6%)	0	0	1 (2.6%)	0.05
	41-50	2 (5.3%)	3 (7.9%)	1 (2.6%)	6 (15.8%)	
	51-60	4 (10.5%)	2 (5.3%)	3 (7.9%)	9 (23.7%)	
	61-70	4 (10.5%)	3 (7.9%)	5 (13.2%)	12 (31.6%)	

	>70	2 (5.3%)	3 (7.9%)	5 (13.2%)	10 (26.3%)	
Gender	Male	6 (15.8%)	9 (23.7%)	7 (18.4%)	22 (57.9%)	0.01
	Female	9 (23.7%)	3 (7.9%)	4 (10.5%)	16 (42.1%)	

Table 1: Demographic and Clinical Characteristics

DR Severity and Systemic Parameters

Severe DR was associated with elevated systolic blood pressure (135.4±18.6 mmHg in PDR vs. 124.3±12.8 mmHg in normal fundus, p=0.03), poorer glycemic control (HbA1c 10.8±2.5% vs. 8.1±1.6%, p<0.001; FBS 167.2±55.3 mg/dL vs. 132.5±34.1 mg/dL, p=0.01; PPBS 245.8±72.9 mg/dL vs. 185.6±58.2 mg/dL, p<0.01), and nephropathy markers (serum creatinine 2.4±1.5 mg/dL vs. 1.2±0.6 mg/dL, p<0.01; blood urea 38.5±18.2 mg/dL vs. 25.4±11.3 mg/dL, p=0.02). [Table 2]

Parameter	Normal Fundus	NPDR	PDR	p-value
Systolic BP (mmHg)	124.3±12.8	130.2±15.4	135.4±18.6	0.03
HbA1c (%)	8.1±1.6	9.5±2.1	10.8±2.5	<0.001
FBS (mg/dL)	132.5±34.1	150.3±45.7	167.2±55.3	0.01
PPBS (mg/dL)	185.6±58.2	215.4±65.1	245.8±72.9	<0.01
Serum Creatinine (mg/dL)	1.2±0.6	1.8±1.0	2.4±1.5	<0.01
Blood Urea (mg/dL)	25.4±11.3	32.1±14.7	38.5±18.2	0.02

Table 2: Association of haematological parameters with DR severity

Cardiac Remodeling

Progressive DR correlated with myocardial remodeling:

- **LVDD**: Increased from 42.5±4.2 mm (normal fundus) to 47.3±6.5 mm (PDR, p=0.01).
- **LVDS**: Rose from 28.3±3.8 mm to 33.2±5.7 mm (p=0.02).
- **LVEF**: Declined from 55.2±6.1% to 43.5±8.9% (p<0.001).
- **LA Diameter**: Increased from 34.6±3.9 mm to 40.1±5.2 mm (p<0.01) (Table 2).

ECG abnormalities were significantly higher in PDR (90%) compared to NPDR (62.5%) and normal fundus (30%, p<0.001). [Table 3]

Parameter	Normal Fundus	NPDR	PDR	p-value
LVDD (mm)	42.5±4.2	45.1±5.3	47.3±6.5	0.01
LVDS (mm)	28.3±3.8	30.7±4.9	33.2±5.7	0.02
LVEF (%)	55.2±6.1	48.9±7.4	43.5±8.9	<0.001
LA Diameter (mm)	34.6±3.9	37.2±4.5	40.1±5.2	<0.01
ECG Abnormalities (%)	30.0	62.5	90.0	<0.001

Table 3: Echo and ECG abnormalities across DR grades

Quality of Life

Quality of life declines step-wise with DR severity, from 'good–excellent' in Mild NPDR to 'very poor' in PDR — a highly significant (p < 0.001) reflection of systemic disease burden.

Grade of DR	Mean KCCQ-12 Score (± SD)	QoL Category	p-value
Mild NPDR	78.6 ± 6.4	Good – Excellent	< 0.001
Moderate NPDR	65.2 ± 7.1	Fair – Good	< 0.001
Severe NPDR	52.8 ± 8.6	Poor – Fair	< 0.001
PDR	38.4 ± 9.2	Very Poor – Poor	< 0.001

Table 4: Correlation of severity of DR with the QoL

Diabetes Duration

Retinopathy, cardiac remodeling, and nephropathy markers all worsen significantly in patients with >10 years of diabetes as shown in table 5.

Parameters		DM < 10 Years	DM > 10 Years	P Value
	Normal	65.2%	12.5%	<0.001*

Retinopathy Severity	NPDR	28.3%	56.3%	
	PDR	6.5%	31.2%	
Cardiac remodeling	LVDD (mm)	42.1 ± 4.0	46.8 ± 5.9	0.002*
	LVEF (%)	54.3 ± 6.2	44.7 ± 8.1	<0.001*
Nephropathy Markers	S. Creatinine (mg/dL)	1.4 ± 0.8	2.3 ± 1.4	0.01*
	Blood Urea (mg/dL)	32.4 ± 15.2	31.9 ± 14.9	0.01*

Table 5: Association of micro and macrovascular complication with duration of DM

DISCUSSION

This study establishes a robust correlation between diabetic retinopathy (DR) severity, myocardial remodelling, and quality of life (QoL) decline in T2DM patients, highlighting shared pathophysiological mechanisms such as oxidative stress, inflammation, endothelial dysfunction, and renin-angiotensin-aldosterone system (RAAS) activation[6,7]. The progressive increase in left ventricular end-diastolic diameter (LVDD), left ventricular systolic diameter (LVDS), and left atrial (LA) diameter, coupled with a significant reduction in left ventricular ejection fraction (LVEF) in severe DR, aligns with findings that diabetes-induced microvascular damage contributes to cardiac remodeling[17]. The high prevalence of ECG abnormalities in proliferative DR (PDR) (90%) corroborates evidence of elevated cardiovascular risk in advanced DR[18].

Systemic parameters, including elevated systolic blood pressure and poor glycemic control (higher HbA1c, fasting blood sugar [FBS], and postprandial blood sugar [PPBS]), worsened with DR severity, consistent with prior studies [14,15]. The concurrent rise in nephropathy markers (serum creatinine, blood urea) with DR progression reflects the systemic nature of diabetic complications, affecting multiple organ systems[16]. The shift to insulin therapy in 75% of PDR patients, compared to 20% in those with normal fundus, indicates the need for intensive glycemic management as DR advances[20].

The significant QoL deterioration with DR progression, as evidenced by the Kansas City Cardiomyopathy Questionnaire (KCCQ-12) scores in Table 4, is driven by visual impairment, cardiac symptoms, and psychological distress. The stepwise decline in KCCQ-12 scores from 'Good–Excellent' in mild NPDR (78.6 ± 6.4) to 'Very Poor–Poor' in PDR (38.4 ± 9.2, $p<0.001$) underscores the compounding burden of visual loss and cardiovascular dysfunction on patients' physical and emotional well-being. These findings align with prior research demonstrating that the combined impact of diabetic complications significantly impairs QoL, emphasizing the need for holistic management strategies[19].

The longer diabetes duration (>10 years) as a critical risk factor for severe DR, cardiac remodeling, and nephropathy emphasizes the cumulative impact of chronic hyperglycemia [21]. These findings position DR as a clinical marker of systemic vascular dysfunction, necessitating integrated ophthalmic and cardiovascular screening.

1. **Mechanistic Insights into Shared Pathways**: The correlation between DR and myocardial remodelling may be driven by advanced glycation end-products (AGEs) and their interaction with receptors (RAGE), which promote fibrosis and vascular stiffness in both retinal and cardiac tissues[22]. Elevated pro-inflammatory markers (e.g., ZAG, RBP-4) in advanced DR suggest a systemic inflammatory state that could exacerbate myocardial fibrosis, as evidenced by increased galectin-3 levels in T2DM patients with left ventricular hypertrophy[23].
2. **Clinical Implications for Risk Stratification**: The predictive value of DR severity for cardiovascular events, as demonstrated by the high prevalence of ECG abnormalities in PDR, suggests that retinal assessments could serve as a non-invasive tool for cardiovascular risk stratification[24]. Integrating retinal screening into cardiovascular protocols could enable earlier detection of subclinical myocardial remodelling, potentially reducing heart failure incidence.
3. **Role of Advanced Imaging**: While echocardiography provided valuable insights, advanced modalities like cardiac magnetic resonance imaging (CMR) with T1 mapping could enhance detection of diffuse myocardial fibrosis in DR patients[25]. Similarly, optical coherence tomography angiography (OCTA) could quantify retinal microvascular changes, offering a more precise correlation with cardiac microvascular dysfunction.

4. **Gender and Age Disparities**: The higher NPDR incidence in males (57.9% vs. 42.1% in females) may reflect differences in treatment adherence or hormonal influences on vascular health[13]. The age-related increase in DR severity, particularly PDR in those >70 years, underscores the role of cumulative hyperglycaemic exposure[12].
5. **Psychosocial and Economic Burden**: The QoL decline in PDR patients, driven by visual loss and cardiac symptoms, is compounded by the economic burden of managing multiple complications[26]. The psychological toll, including anxiety and depression, may impair glycaemic control, creating a vicious cycle.
6. **Research Gaps and Future Directions**: The cross-sectional design limits causal inferences, necessitating longitudinal studies. Exploring biomarkers like NT-proBNP or galectin-3 could improve early detection of cardiac remodeling in DR patients[23]. Investigating the impact of novel therapies, such as SGLT2 inhibitors, on both DR and cardiac outcomes could further refine management strategies.

Limitations

Small, regionally and socioeconomically homogeneous sample limits generalizability. Lack of lifestyle data like diet, exercise, and medication adherence affects understanding of disease progression. Results are limited to one hospital; multicentre studies would enhance data diversity and strength. Cross-Sectional study design limits ability to infer causality between DR and myocardial remodelling; longitudinal studies needed.

CONCLUSION

DR severity in T2DM patients is strongly associated with myocardial remodeling, increased cardiovascular risk, and reduced QoL. Severe DR, particularly PDR, serves as a clinical marker for cardiac dysfunction, emphasizing the need for integrated ophthalmic and cardiovascular screening. Early detection, aggressive glycemic control, and multidisciplinary care involving endocrinologists, cardiologists, and ophthalmologists are essential to mitigate complications. Comprehensive support addressing physical and psychological needs is critical to improving QoL. Future longitudinal studies with larger, diverse cohorts and advanced imaging are warranted to confirm causality and refine management strategies.

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